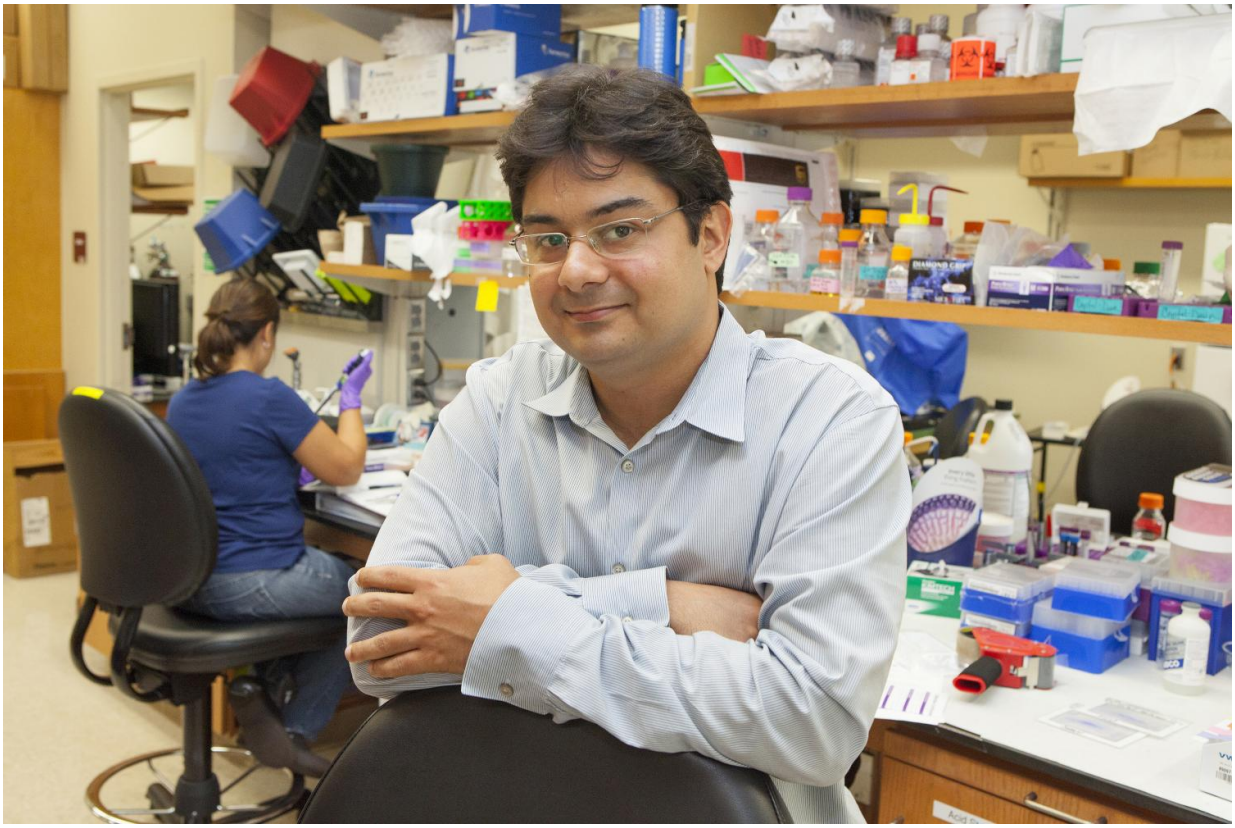
Researchers target on-off switch of cardiac contraction

May 11 2016



Jose Pinto is a researcher in the Florida State University College of Medicine. Credit: FSU Photography Services/Bill Lax

Calcium is most associated with bones, but it's also a key player in the heart.

When calcium binds to a certain protein in heart cells, it triggers muscle contraction. Too much calcium causes stronger heartbeats; too little calcium leads to weaker beats.

Jose Pinto, a researcher in the Florida State University College of Medicine, will spend the next five years studying how the heart's calcium is regulated and how to correct a calcium imbalance using an inside approach in the cardiac cell. The National Institutes of Health has awarded him a $1.8 million grant, which will allow him to continue the work he began last year with funds from an American Heart Association award.

"The concept and the aims are similar, but we are now going much deeper into the molecular mechanisms," said Pinto, an assistant professor in the Department of Biomedical Sciences.

The long-term goal is to identify components inside the cardiac cell that are involved with the development of cardiomyopathies—diseases that can lead to heart failure—and open the door for more effective treatment strategies inside the cell.

Troponin C is a gene that Pinto calls "the calcium sensor of the heart," the on-off switch controlling contractions in the heart. His team is working to demonstrate that mutations in that gene cause the heart to pump blood in one of two harmful ways. One results in hypertrophic cardiomyopathy, where the heart becomes abnormally thick and initially pumps more forcefully. The other is dilated cardiomyopathy, where the pumping chamber stretches and the walls become thinner—and thus pumps more weakly.

"In the hypertrophic heart, it can be compared to a dimmer switch; you turn it only a little bit and the lights come on—it's hypersensitive to calcium," Pinto said. "In the dilated cardiomyopathy, it's like you switch,

switch, switch, switch, and the lights never come on."

Once he successfully demonstrates Troponin C's role, he aims to focus on a possible treatment of hypertrophic cardiomyopathy using an enzyme found inside heart cells.

"The beautiful thing about the enzyme we are targeting is that it has been shown to be only present in the heart," Pinto said. "The main problem with most drugs developed to target enzymes is that they affect other cells, healthy cells, and the patient experiences unwanted side effects."

Pinto believes that targeting hypertrophic cardiomyopathy from within the heart cells may help prevent those unwanted side effects.

"If your heart is working above the normal range all the time, that is going to lead to problems," Pinto said. "The unique thing about our project is that we're going to be studying what the deletion of this enzyme will do in the heart after the disease has already started. We are looking at the reversal of the disease: Can we do something to bring the heart back to normal?"

Provided by Florida State University

Citation: Researchers target on-off switch of cardiac contraction (2016, May 11) retrieved 29 April 2023 from https://medicalxpress.com/news/2016-05-on-off-cardiac.html

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